Art. XVIII.—*Paralysis in Horses and in Cows due to the Ingestion of Fodder.*

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[Read 12th September, 1912].

For many years a form of progressive paralysis in cattle, evinced primarily by an inability to ingest food, associated with absence of rumination, more or less rapid emaciation and early death, has been recorded as prevalent in Victoria, South Australia, and even in certain districts of Tasmania.

Many articles have appeared in the lay and agricultural press dealing chiefly with the symptoms, the seasonable occurrence, the nature of the food supply, and, to some extent, the *post-mortem* appearances, but little systematic enquiry into the etiology seems to have been attempted.

So far as I am aware, the first accurate and scientific description of the disease was published in the "Australasian Farm and Home," in April, 1896, by my colleague, Dr. W. T. Kendall, and his account of the symptoms is so careful and concise, that no one having perused it could fail to recognise a typical case. Under the head of "*post-mortem* appearances," it is worthy of note that Dr. Kendall states regarding those cases he examined :—"The supposed impaction of the omasum or third stomach to which the mortality has been entirely attributed, did not exist."

Dr. S. S. Cameron, in Victoria, who gives in various articles a definite account of the symptoms, concludes that the condition is a paralysis of the 9th and 10th cranial nerve due either to some form of mould (fungus) poisoning or solely to the non-succulent and innutritious nature of the food supply, either of which he considers a useful hypothesis for future work. At a later date I understand he suggested the term "Impaction Paralysis," on account of the impaction of the rumen, which, in his experience, was almost invariably present. He appears to consider that the diseases known popularly as "Stomach Staggers," "Winton Disease," "Cripples," etc., are one and the same.

In South Australia Mr. J. Desmond has given much attention to the condition. (See report to the Hon. Minister for Agriculture, and Veterinary Journal, Vol. 66, No. 417, page 161.) In his first

reports he also considered the disease due to impaction of the omasum and termed it "Dry Bible," but in his last report he claimed to have found the specific bacterium. A description of this organism, and details of his experiments, however, have never been published, hence they cannot be dealt with here.

Dr. Wilmot, in Tasmania, recently in a special report claimed that the disease is really the same as "Louping Ill" of sheep in Great Britain, but his evidence is, to say the least, very unsatisfactory.

Prior to joining the Melbourne University in 1909, I had naturally been aware of the existence of this disease, and hoped to have an early opportunity of observing definite cases, if not of making a thorough investigation of the whole condition. It was only, however, in the late summer months of 1911 that I was able to examine for myself definite cases in cattle, cases that everyone was agreed were typical, and this through the courtesy of Mr. Humm, G.M.V.C., Warrnambool.

During April, 1909, three cows were sent to me by the Department of Agriculture's officials, but none were typical. Indeed, some of those who had had considerable experience of this form of paralysis in cows considered they were not cases at all. One certainly suffered from acute broncho-pneumonia, and died within 36 hours after admission to the hospital, but the other two I now consider were affected with a mild form of the disease.

That the symptoms are frequently very indefinite is proved by the fact that many cases have been reported to me, by laymen chiefly, which proved on examination to be of quite a different nature to the disease under review. For example, one outbreak of pleuro-pneumonia was originally reported both to the Department of Agriculture and to myself, as a severe outbreak of this disease.

The paralysis of the horse, although due to the same cause as that of the cow, is of an entirely different character so far as the muscles affected are concerned.

The disease has been little observed in Victoria, judging by available literature, but in South Australia it appears to be fairly common, and often assumes the character of a severe endemic. It has been carefully studied by Mr. J. F. McEachran, M.R.C.V.S., who, both in correspondence and in consultation, has afforded me valuable information regarding the disease.

Until recently, and indeed until the experiments recorded later were being conducted, the connection between the disease in the cow and that in the horse was not suspected. This is not surprising when the symptoms are compared, especially in view of the fact

that while occasionally on farms in South Australia the disease may occur in both cattle and horses, it more frequently happens that the one or the other class of animal is alone affected. Why this is so will, of course, be a matter for further investigation.

Though much has been written regarding the symptoms observed, especially in the cow, and to some extent in the horse, in this article I propose to confine myself to recording what I have personally observed and ascertained from owners of the affected stock.

Symptoms in Cow.

Usually the first symptom of illness observed by the dairyman is either a severe diminution, or almost complete cessation, of the milk supply in a cow which at the previous milking has yielded her normal quantity.

It is more than probable, however, that a careful observer might detect some symptom prior to this, especially if close attention were paid to the animal when feeding or at rest; but in any case, such abnormality is liable to be overlooked until the milk yield is affected. Even then other symptoms are by no means very definite. Rumination is often suspended, but this is not unusual in many cattle diseases. Often it is intermittent. The most definite and probably most characteristic symptom at this stage is a peculiar, slow and very persistent movement of the jaw, not the ordinary movement which occurs during either chewing food or cud, but a slow up and down movement, with little, and often no, lateral movement of the lower jaw. This movement seems to be almost involuntary; it will cease when the animal's attention is distracted, and is resumed again later on. The head is held somewhat straighter than normal, and this becomes more pronounced during the progress of the disease. The expression is peculiar, it may almost be termed wistful and enquiring. Inattentiveness to surroundings is observed, and the animal is generally seen apart from the herd. No attempt to eat or drink is made, but whether at this stage because the animal knows she can only chew and swallow, if at all, with difficulty, or through experience of ineffectual attempts, or through intuition, I have not been able to determine. There is, however, from the first, I think, but little appetite, though it may not have entirely disappeared. In the very mild cases, or cases which progress slowly, a little water may be swallowed, but with great difficulty. In one instance I observed a cow hold her mouth in a bucketful of water for ten minutes, and only succeed in drinking half the contents. On another occasion I observed a cow which

we had driven into a waterhole, hold her mouth in the water for almost fifteen minutes. In both these cases some time elapsed before any liquid was swallowed, as observed by carefully watching the movements of the oesophagus, while but a small quantity was swallowed at a time, and at infrequent intervals.

From the beginning, prehension of food, even cut green fodder, is difficult, and soon becomes impossible, even though the animal may be obviously anxious to partake of the material. When it is possible to seize the food, mastication cannot be performed. An attempt is made to chew a blade of cabbage, a stalk of sow thistle, some green clover, or such material introduced into the mouth, but the process resembles more the action of a human being sucking a sweet than the normal mastication of a cow. The buccal muscles do not seem to come properly into action, and there appears to be inability to properly direct the tongue. During this attempt to masticate there is considerable salivation. Often such partially or very slightly chewed material which has been introduced into the mouth will be dropped, and if it is not, it rarely gets beyond the base of the tongue just in front of the velum. Indeed, in nearly all cases it has been observed by most writers that a quantity of partially chewed food is almost invariably found situated there on *post-mortem* examination. In two cattle dead of the disease, or what was probably the same disease in Tasmania, which I had an opportunity of examining *post-mortem* (although unfortunately dead nearly 48 hours), this plugging of the base of the tongue by food was very definite.

But the difficulty in swallowing does not cease here; it is evident that even the oesophagus is in a semi-paralysed condition, for in one case where with considerable patience and persistence the patient had slowly and with some distress succeeded in swallowing a little fresh grass, I found on *post-mortem* examination a few hours later, the ingesta lying along the lumen of the intra-thoracic portion of the oesophagus like a loosely twisted green rope.

As the disease progresses, the champing of the jaws becomes more noticeable, the tongue is frequently protruded as if to make an attempt to lick the dry muzzle, but without success, and may remain so protruded for several seconds or even longer. Salivation becomes more profuse, there being a constant dribbling from the mouth of saliva, which is often ropy in consistence. There is frequently an intermittent mucoid discharge from the nostrils, which is probably saliva which has been only partially swallowed and returned *per* nasal chamber. The expression becomes more wistful and the head more straightly held A slight swaying of the body,

accompanied by twitching of muscles, appears, but not invariably, and seems to indicate that the animal is afraid of falling, and even of lying down, in case she cannot rise again. Cases may be found recumbent, but generally in the early stages; when symptoms are very definite the rule is for the patient to stand quietly, even if placed in a loose box. In a box the head may be often found pressed into the corner, as if a partial support were given thereby. If forcibly moved, the gait is more or less staggering, and the desire is to move only in a straight line. Respirations are normal, except when disturbed. The temperature remains normal, though occasionally reaching 103 deg. F., particularly when exposed to the sun. The faeces are scanty but not abnormal. Micturition is generally infrequent, but may be the reverse, and may be accompanied by evidences of pain. At times injection of the visible mucosae may be present. Occasionally some evidence of internal colicy pains is observed, but this is not present in all cases. More or less rapidly, the animal becomes weaker and weaker, chiefly from lack of food, and, especially in animals unhoused, from lack of water. Sooner or later the patient lies or falls down, and can be got to rise with difficulty, or not at all. Gradually a condition of semi-coma ensues, with complete coma and death resulting. Usually the course of the disease lasts from two to four days, but may linger for a week after symptoms are observed. Occasionally, it is said, cases recover, but I believe no immunity is attained against a recurrent attack the next year. I feel certain that were sick animals housed and so kept away from the sun's rays, and especially were they coaxed to drink from the beginning, they would survive much longer. Consequently chances of recovery would be much greater.

The most characteristic symptoms I consider to be the champing of lower jaw, the inability to chew and swallow, the salivation, the expression, the attitude of head; in other words, partial or complete paralysis of the powers of prehension, mastication, and deglutition. The disease is rarely, if ever, seen in animals below two years of age, and apparently never in calves. In Victoria it does not often attack steers, or at all events is rarely observed in that class of stock, although bullocks are not infrequently attacked in Tasmania.

The season of the year when the disease prevails is the dry summer months, especially January and February, though cases may occur earlier or later. For this reason, the dry, often innutritious, nature of the food supply is considered by some as being the primary cause of the impaction and paralysis of the rumen.

Post-mortem Appearances.

There are no pathological macroscopic changes which are constant or in any way characteristic.

The so-called impaction of the omasum, with dry ingesta, which led to the ridiculous term "Dry Bible," observed by some and considered the *causa causans* of the disease, is not constant, and is not greater than may be seen often in long travelled bullocks killed at abattoirs, and is not surprising considering the want of food and water, which may have been for a longer period than generally estimated. The brain and spinal cord are quite normal. The meninges may show slight injections, and while in some cases there has been an abnormal quantity of clear meningeal fluid present, though not sufficient to cause the symptoms noted. in others there was no such excess. The buccal cavity and pharynx are generally quite normal, and in only one instance have I observed congestion of the pharyngeal mucosa. Contrary to some observers, I have never found any food in the pharynx proper, but almost invariably there is to be found a pledget of partially-chewed material situated between the base of the tongue and the soft palate. In addition to this, one may find partially chewed or even unchewed food within the oesophagus, especially the intrathoracic portion. The rumen generally contains matted masses of food, though in one case which had been frequently placed by me in a pond, and had with difficulty by very persistent effort drunk a quantity of water, it was found, when the animal was slaughtered soon afterwards, that this water was almost entirely within the rumen. The abomasum is invariably empty; the small intestines are in a catarrhal condition, and contain more or less milky mucoid material, particularly the duodenum. The large intestines contain normal contents, though small in quantity. All other organs are normal, including the bladder, which is generally empty.

Bacteriological Investigation.

Microscopical examinations of blood and other body fluids have always given negative results. Injection subcutaneous, intraperitoneal, and intravenous of other cows with blood, spinal fluid, and even with emulsion of brain and spinal cord (brought to the laboratry preserved in glycerine and normal saline) have invariably given negative results. A cow drenched with a quantity of the catarrhal material found in the small intestines remained normal. The conclusions to be drawn from these experiments are that the

disease is not due to any living organism visible or ultra-visible. Naturally, however, such were the first experiments to be undertaken, especially in view of the discovery of an ultra-visible virus being the cause of human polio-myelitis by Levaditi and Flexner.

Symptoms in Horse.

Instead of localised paralysis as in the cow, the symptoms in the horse are those of a general paralysis. Premonitory symptoms consist, at most, of inappetance. Frequently the animal is not observed ill till seen lying on the side, unable to get up even with assistance, and there may be evidence of intermittent colicy pain. In some cases the first symptoms are those of hyper-acute and sudden attack of colic, from which apparent recovery may occur, to be succeeded, however, by complete motor paralysis of the limbs. Often a peculiar paddling of one or more limbs is observed while the animal lies recumbent. In some cases the patient lies apparently quite quiet for prolonged periods, in others colicy griping pains of the intestines appear to be frequent, judging by the attitude, particularly the turning of the head to the flank. Sensation in the early stage is not lost, as can be demonstrated by pricking the skin, when the muscles of the flank or shoulder will twitch, but the limb cannot be moved voluntarily. The paddling movement appears to be entirely involuntary.

In the early stage the animal retains consciousness, though in the acute cases this soon gives way to semi-coma. The pupils are dilated, and the expression is startled-looking. The temperature remains normal, or it may be slightly above normal. The pulse is weak and rapid. The visible mucosae are slightly injected. Perspiration is often profuse, and may be patchy.

Depending upon the acuteness of the case, semi-coma, succeeded by coma, occurs, the paddling movements cease, and death may take place within 12 to 24 hours from the onset of the symptoms, or in subacute and chronic cases, the animal may linger for several days. Death is usually preceded by a violent paroxysm.

These are the symptoms as observed here in a number of both accidentally and deliberately produced cases.

Post-mortem Appearances.

Skin and subcutaneous bruises are common, but are only bed sores.

In two cases patches of semi-gelatinous, straw-coloured oedema were present in the peri-pharyngeal connective tissue, but

were absent in others. The brain and spinal cord show no lesions beyond at times a slight injection of the ventricles. The meninges are generally normal, but in one naturally acquired case, and in one deliberately conferred case, there was an abnormal quantity of clear cerebro-spinal fluid present, especially around the medulla.

The lungs in very acute cases show passive congestion; the pericardium contains a varying, though small, quantity of clear serosity; the heart shows sub-epi- and sub-endo-cardial petechiae; the blood coagulates readily with a firm clot and clear serum. The stomach is generally normal, in only one case a few ecchymoses being seen near the pylorus. The small intestines are invariably in a muco-catarrhal condition with much serous effusion, coagulable on exposure, and mixed with flocculi of shed epithelium. This is found especially in the anterior part of the canal. In one case small necrotic patches of the duodenal mucosa were observed. The large intestines are generally tympanitic, but contain normal ingesta. One case showed a large area of congestion and many petechiae of the colon. The liver and kidneys are slightly congested, and show cloudy swelling. Otherwise the organs are normal.

Bacteriological Examination.

Microscopical examination of the blood and other fluids, such as cerebro-spinal fluid and pericardial serosity, as well as tissues, gave always negative results. Blood secured from the heart and large vessels in sterile pipettes and tubes immediately after death coagulated readily, and remained sterile even at blood heat. Intravenous inoculation with such material gave negative results, so that a living virus as the exciting cause was excluded, so far as such experiments could determine. Fortunately the fodder which had been fed to horses that had succumbed in an outbreak detailed below had been secured by me, and tests with this gave interesting and surprising results.

Observations on outbreak of Horse Paralysis in a Melbourne stable.

In the beginning of November, 1911, I was advised by Mr. W. A Kendall, C.M.V.C., of a serious outbreak of disease affecting three horses belonging to a dealer in one of the suburbs. The three horses, of a very good draught type, became affected almost simultaneously, and succumbed within 24 hours, in spite of treatment. Mr. Ken-

dall's account of the symptoms, and the observations I was able to make through his courtesy, tallied generally with the symptoms recorded above, as did the *post-mortem* examinations, at only one of which I was able to be present and personally secure specimens. The cases did not seem to be those of ordinary poison, and chemical examination of the stomach contents and of the fodder gave negative results. Fortunately, however, I had a quantity of the fodder, consisting of ordinary mixed chaff, with bran and oats, all apparently of good quality, sent to the laboratory for experimental purposes. The material collected was kept separate, and labelled according to the place of collection. These samples were :—

A. From the mangers of the horses that had died.

B. From the nosebags of those horses.

C. From the loft near the chute.

D. From the bulk fodder in the loft.

(N.B.—C and D were similar, with the exception that D was mixed later by a few days than C.)

No unmixed feed remained on the premises.

With the above feed, certain experiments were conducted, but before detailing these it is necessary to record what may well be termed an unpremeditated experiment.

When the first horse died, the knacker was instructed to come for the carcase, and his man arrived with the cart sometime before Mr. Kendall had finished his *post-mortem* examination. While waiting, he espied a nose bag half-full of feed hanging on a peg, and fixed it on his horse's head. The knacker's horse, although fed as usual only a few hours previously, partook of the chaff in the nose bag readily enough, and in due course left. Five days later Mr. Kendall was called in to see this animal, and found him suffering from symptoms exactly similar to those manifested by the original cases. Death occurred within 12 hours of the first symptoms being observed. The carcase was brought to my laboratory, and *post-mortem* examination showed conditions similar to the others. This horse had received no more than a few pounds of fodder, and no symptoms were declared for at least five days. Meanwhile our experiments were in progress.

Horse E. 11, an aged animal, in fair condition, was fed about 5 p.m. with 5 lbs. of chaff from the mangers. This he ate readily enough. For the next five days he received a daily ration of 15 lbs. of chaff from D, the bulk feed. On the morning of the 6th day he ate his food as usual. In the evening at 5 p.m. he was again fed,

and at 6.30 p.m. it was observed he had not eaten all the food, though otherwise he appeared normal. At 9.15 p.m. (or a little over five days after the first feed had been given) the animal was found lying down in the box, evidently suffering from abdominal pain. Temperature 102 deg. F. Pulse full, but weak and somewhat irregular. Respiration normal, eyes bright with pupils dilated. When disturbed, he rose and soon afterwards ate a little food. At 10.45 p.m. he was again found lying down, groaning with colicy spasms, and struggling. He tried to get up, but was unsuccessful. He continued to struggle violently for some minutes, but later settled down. Pulse much accelerated, but gradually slowed.

At 11.45 p.m. there was another paroxysm similar to last, but struggling was practically confined to continual banging of the head on the floor, and paddling motion of hind limbs. Gradually he quietened down, and half-an-hour later manifested little tendency to struggle when touched, but remained sensitive to all sounds and skin irritation. Temperature 100.2 deg.

At 12.45 a.m. another paroxysm of struggling occurred, so the animal was given morphine subcutaneously, and left for the night, having been well bedded down.

Next morning the animal was found to have knocked himself about a good deal during the night. Throughout the day he lay quiet for the most part, but off and on there were periods of struggling. Temperature did not rise, but respirations became more laboured, though hypersensitiveness was still marked, until semi-coma ensued shortly before death. The animal died at 5 p.m., 20 hours after having been first noticed lying down, having struggled a good deal during the last hour.

Post-mortem Appearances.—Similar to cases at stable from which fodder was obtained, but less injection of the vessels, little catarrh of small intestine, no oedema around pharnyx, and but slight excess of subdural fluid.

The feed left in the manger of this horse (about 6 lbs.) was divided between two sheep. They ate it readily enough, and remained normal during the several weeks they were under observation.

Pony E. 12 and Cow E. 1.—The remainder of the feed from the manger A was fed to pony E. 12, which received 3 lbs., and to a cow 4 years old, in good condition, which received 9 lbs. For the next week ordinary fodder, as given to other animals at the

laboratory stables, was supplied to these animals. Then of the fodder found in the nose bag (B), 2 lbs. was given to the pony, and 5 lbs. to the cow. This was an error, for it was intended by me they should be fed thereafter with material C, from near the chute in the loft. This material was used subsequently, the pony receiving 3 lbs. and the cow 12 lbs. per day.

It may be here observed that the division of the feed was purely arbitrary, for we had no reason to believe either from the owner's statements or from observation that there was any material difference. The lots were kept separate, so that if necessary any definite poison might be tested for in each. The feeding with the bulk material was adopted because the pony had not evidenced any illness, and there appeared no reason for keeping the material for too great a length of time, besides which, it saved our own feed bill.

The result of the experiment was very interesting and extremely instructive.

The pony ate well and appeared normal till the thirteenth day after the first feed, when inappetance appeared, the morning feed being refused. In a few hours a fit of colic occurred, which soon passed off. These attacks recurred at intervals, till later in the day, when they ceased, though the animal still remained dull, listless, and refusing food and water.

Next morning the pony was found lying on his side, the limbs straight out, without making any effort to get up. Consciousness was definite, the head being partially raised from time to time. No evidence of pain. No desire for food or water. Skin of limbs sensitive, as shown by twitching of body muscles on pricking, but otherwise no reflex action or voluntary action was observed. Temperature 100.4 deg. to 101.2 deg. Pulse and respirations normal. No faeces or urine passed. The following or second day condition was much the same. Consciousness remained complete, the pony neighing on hearing another horse pass by the box. When the head was held up, he drank fairly readily, but refused food; he showed paddling of limbs at intervals, respirations were increased, and towards night, the breath was offensive, mouth was clammy, no faeces or urine had been passed, catheter showing comparatively little of latter in bladder, and that of normal character; the pulse was slow and full. During the next two days, little or no change was observed. Bed sores became extensive in spite of frequent turning. A little dung, dry in character, was passed occasionally. Pulse increased to 80 per minute. Temperature remained normal, and breathing was slightly accelerated. Consciousness was still

retained, the chief symptom being inability to move the limbs except by the involuntary paddling referred to. At the end of the fourth day no improvement and no aggravation in general symptoms having taken place, except increasing weakness, because of the bedsores and the absence of feeding, the pony was killed.

Post-mortem examination was much the same in results as in previous cases, with the exception of some areas of broncho-pneumonia.

The experimental cow gradually became affected with tongue, buccal, and pharyngeal paralysis. This cow made an admirable subject for experiment. For six months she had been kept (in consequence of another experiment) in a loose box along with a yearling calf, and in the next box another cow had been kept for the same period also with a yearling calf. During that period the four animals had all been fed on the same kind of fodder, i.e., dry chaff, hay, etc., with no green feed, or exercise. Although, as will be seen, the cow fed with the suspected chaff became affected with the paralysis, the other cow and calf remained normal, and continued to do so while fed on ordinary chaff, till killed two months later. Her own companion had been destroyed prior to commencing the feeding experiment. No symptoms of any abnormality whatever were detected till the thirteenth day after receiving the first feed, or the sixth day of continuous feeding on suspected fodder. Then the cow was noticed to be less keen for food, and next day seemed to chew each mouthful for a much longer period than usual, without making any attempt meanwhile to swallow. Although the day was warm, little or no water was drunk. It is safe to say that had a careful watch not been kept for some such evidence of disturbance, it would have been overlooked by the attendant. On the third day of sickness the condition was much the same, but a thin trickle of saliva ran from the mouth, and some mucoid looking discharge was present in the nostrils. The fourth day brought improvement, and the cow seemed practically normal, but on the fifth day the aimless chewing and slight salivation had returned, while the expression was somewhat strained. On the sixth day, the symptoms were for the first time very suggestive of the cattle paralysis. Feed was refused; the head was held straighter than normal, the eyes appeared staring, champing of the jaws with dribbling of saliva was constant, occasionally the tongue protruded in an ineffective way, but seldom was an attempt made to lick the muzzle. On the seventh and eighth days the symptoms were aggravated. Drinking was done slowly and with much difficulty. On the ninth day the condition was much

the same, the animal continuously standing, staring unseeingly in front of her, the salivation and movement of the jaw in the characteristic way being almost incessant. A blade of cabbage was picked up and sucked laboriously, being finally rejected in an unchewed condition. It was obviously impossible for her to get the food between the teeth or backward into the fauces. After this she refused cabbage, but attempted a little grass with the same result, and the next time grass was offered, she refused that. Evidently therefore, the appetite was still present, but she remembered her inability to chew or swallow certain materials. Rumination had ceased to be observed for several days. On this ninth day, also, the rumen was slighly tympanitic, and occasionally eructations of gas and fluid occurred, the latter passing down the nostrils.

For the next three days there was little change except that the animal became gradually weaker and poorer in condition; as always, a little faeces and urine were passed, but no food or water was taken. The temperature ranged between 100 and 102 deg.

During this time several veterinarians and others who have had much experience of the cattle paralysis as it naturally occurs, saw the case, and unanimously confirmed my diagnosis that it was a typical, though not acute, case of that disease.

During the night of the twelfth day after the first symptoms were manifested, she gave birth to a fully-developed, healthy calf, but was herself found prostrate and unable to rise in the morning. As she had eaten practically nothing for five days, and very little for a week before that, this weakness was perhaps not surprising. Nothing apparently was to be gained by attempting treatment, so she was killed.

Post-mortem examination showed no abnormality that could be considered in any way pathological or associated with the symptoms, and no excess of cerebro-spinal fluid.

Horse E. 13, aged, and in comparatively poor condition. It was fed for twelve days with 10 lbs. per day of bulk chaff from the loft, and was run in a small bare paddock. The supply of chaff being finished, he was then placed on the ordinary fodder of the laboratory. No evidence of any illness was manifested until sixteen days after the experiment commenced, when he refused his feed, but otherwise did not appear ill. The following day he was found lying on his side, presenting all the symptoms of paralysis observed in the pony, and in the other experimental horse. The next day (Christmas Eve) no change having occurred, and it being evident he might live for several days without any material benefit being derived from studying the case, the animal was slaughtered.

Post-mortem examination was as in other cases.

Summarised, the results of these feeding experiments are as follow:—

(1) *Knacker's Horse.*—Fed with quantity of chaff (B) left in a nose bag, probably not more than 6 to 8 lbs. altogether. *Result:*—In five days symptoms developed, characteristic in every way of the disease as it affected the merchant's three horses, and death occurred under 24 hours later. Other horses belonging to the same owner remained normal.

(2) *Experimental Horse E. 11.*—Fed daily with chaff A removed from the mangers of the horses that died, this being the last feed supplied to those horses, and undoubtedly of the same character as that in the nose bags. *Result:*—Colicy symptoms were exhibited almost precisely five days after first feed was eaten, and three hours later the characteristic symptoms were fully developed, and death occurred seventeen hours later.

The total quantity of feed consumed by this horse was 74 lbs.

(3) *Experimental Horse E. 12.* a pony, fed with 3 lbs. of feed from manger. A week later, no symptoms having developed, it was fed with 2 lbs. from nose bags (B), then fed with 3 lbs. per day of material in loft near chute (C), 33 lbs. of this being eaten altogether up to the time he was observed off his feed. In all, this pony ate 5 lbs. of what had been proved to be poisonous fodder, and 33 lbs. of suspected material, or 38 lbs. altogether. *Result* was that refusal of any food was observed twenty days after first feed, and thirteen days after continuous feeding commenced. This was followed by colicy pains, and later by definite paralysis. In this case, however, the disease was not early fatal, the animal being killed four days after illness commenced, on account of refusal to feed, bed sores, etc.

(4) *Experimental Horse E. 13.*—Fed with chaff (D) from bulk in loft, said to have been mixed a week after that in the manger and nose bags, receiving at the rate of 10 lbs. per day for twelve days, during which time no evidence of illness was manifested, being then placed on the ordinary chaff of the Institute. *Result:*—Fourteen days after feeding commenced, and four days after last feeding of chaff from the merchant's loft he refused food, and next day was found down, and paralysed. The animal was killed 24 hours later, it being evidently a sub-acute case, and the Christmas holidays being in course.

(5) *Experimental Cow.*—Fed simultaneously with pony. Received first 9 lbs. of chaff from manger. A week later received 5 lbs. chaff from nose bags, and thereafter fed daily with 12 lbs. chaff from loft

near chute: *Result:*—On the thirteenth day after first feed, some inappetance was observed, but not marked. Gradually symptoms of buccal, pharyngeal, and to some extent lingual paralysis became manifested; she was killed when almost *in extremis* after having given birth to a healthy calf, and after having eaten and drunk practically nothing for the previous five days, thirteen days after the first symptoms of illness appeared, and twenty-six days after the feeding experiment commenced. In all 88 lbs. of the contaminated or poisonous fodder was eaten by this animal.

It becomes obvious from a consideration of the results of these feeding experiments that the fodder, consisting apparently of ordinary chaffed oaten hay, with the addition of a slight percentage of oats, and of bran, contained some element which was the cause of the fatal results. What this element was, whether some native or other weed, or a fungus (or other) disease of some of the constituent plants, there was no indication, and examination of the cut fodder was unsatisfactory on these points. That the horse paralysis in South Australia, where it has been most observed, is frequently found associated with feeding on musty fodder, would seem to indicate the latter hypothesis; but the fact that this is not invariably the case is against the idea of ordinary moulds being a cause. Again, it must be remembered that if the cause be of a fungoid nature, it is one that does not deleteriously affect the fodder to such an extent as to render it unpalatable or even suspicious to the horse or cow, not to mention ourselves, by any standard we can at present adopt. Further, while I have not heard of any cases of horse paralysis where the animals were not, at least partially, fed on dry fodder such as hay, chaff, etc., cattle paralysis frequently occurs where animals are feeding solely on natural pasturage. The most reasonable hypothesis under the circumstances, is, therefore, that there is some plant which horses are able to avoid while it is growing naturally, but which even then many cattle cannot or do not avoid; a plant also that even the horse does not detect when mixed with other ripe or dried plants, especially if chaffed and mixed.

But again, whether fungoid or herbal in origin, it cannot be gainsaid that the essential cause of the paralysis is some powerful poison of certain cells in the central nervous system. The chemical nature of that poison cannot as yet be even assumed, for we know of no poison alkaloidal or glucosidal in nature, which would have such a latent period before symptoms were produced. In fact, such poisons would be eliminated almost entirely from the system within 24 to 48 hours, provided no physiological effects were produced. In

the case of the two first experimental horses and of the cow, a cumulative effect of the poison might be inferred, but the experience with the knacker's horse, which received only one feed, and with experimental horse E. 13 (the former of which showed symptoms only five days after ingestion of a small quantity of the abnormal fodder, the latter four days after feeding on this was discontinued) negative such an assumption being regarded as a complete explanation. The best analogue of this poison, whatever may be its nature and source, is the toxin of tetanus, which has a so-called period of incubation of four to five days, due to the time taken in reaching the cells of the central nervous system *via* the nerves, instead of by the blood stream. The Rabies virus offers to some extent another analogy, but there it is the living entity that travels along the nerves, and probably liberates the toxin *in situ*.

At all events the whole circumstances seem to indicate the study of the native flora in the affected districts as a field of research offering the most fruitful possibilities. Had the fodder with which I experimented been uncut, I should have had the component plants of a large quantity segregated after the first two experiments, and conducted feeding experiments with each lot. As this was not possible, I requested the Hon. G. Graham, the Minister of Agriculture, to have a botanical survey made by the Government Botanist of the farm in the Western district whereon I had seen the most typical cases of cattle paralysis, to which he kindly consented. This was done, and I append a copy of the report which I received.

The native plants should be secured in some quantity; if necessary they should be artificially cultivated, especially the senecios, pimpernels, isotomas and lobelias, and thorough tests made with each on both horses and cattle.

Mr. McEachran, at my suggestion, is proposing to carry out, at the first opportunity, certain experiments with dry unchaffed fodder, where there has been an epidemic, the endeavour being to segregate the different plants, and carry out feeding experiments with each.

So far as treatment is concerned, I am sure efficacy will only be attained by a study of the poison after it has been isolated, or at least after its origin has been discovered, and meanwhile all endeavours should be directed towards that end.

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APPENDIX.

Report by Professor A. J. Ewart, Government Botanist, on plants found growing on the property referred to in text.

The following is the detailed list of the plants collected :—

Gramineae (Grasses).

Holcus lanatus, L. "Yorkshire Fog Grass." Alien.
Hordeum murinum, L. "Barley Grass." Alien.
Deyeuxia Forsteri Kunth. "Tooth Bent Grass." Native.
Poa caespitosa, G. Forst. "Tufted Meadow Grass." Native.
Lolium perenne, L. "Perennial Rye Grass." Alien.
Briza minor, L. "Lesser Quaking Grass." Alien.
Aira caryophyllea, L. "Silvery Hair Grass." Alien.
Agropyrum scabrum, Pal. "Common Wheat Grass." Native.
Pentapogon Billardieri, R. Br. "Five-awned Spear Grass. Native.
Anthistiria ciliata, L. "Kangaroo Grass." Native.

Cyperaceae, Juncaceae (Sedges and Rushes).

Carex Gunniana, Boot. "Green Sedge." Native.
Carex paniculata, L. "Panicle Sedge." Native.
Heleocharis acuta, R. Br. "Common Spike Rush." Native.
Juncus bufonius, L. "Toad Rush." Native.
Juncus pallidus, R. Br. "Pale Rush." Native.
Juncus planifolius, R. Br. "Broad-leaved Rush." Native.
Juncus communis, E. Mey. "Common Rush." Native.
Juncus prismatocarpus, R. Br. "Branching Rush." Native.

LEGUMINOSAE.

Trifolium resupinatum, L. "Annual Strawberry Clover." Alien.

Trifolium fragiferum, L. "Perennial Strawberry Clover." Alien.

Trifolium minus, Rel. "Slender Clover." Alien.

Trifolium repens, L. "White or Dutch Clover." Alien.

Acacia mollissima, Willd. "Late Black Wattle." Native.

Acacia melanoxylon, R. Br. "Blackwood." Native.

Psoralea parva, F.v.M. "Small Scurfy Pea." Native.

COMPOSITAE.

Carduus lanceolatus, Scop. "Spear Thistle." Alien. Proclaimed.

Carduus pycnocephalus, Jacq. "Shore Thistle." Alien. Proclaimed.

Carduus Marianus, L. "Spotted Thistle." Alien. Proclaimed.

Cryptostemma calendulacea, R. Br. "Cape Weed." Alien. Proclaimed.

Hypochaeris radicata, L. "Flat Weed." Alien.

Anthemis nobilis, L. "Common chamomile." Alien.

Senecio lautus, Soland. Native.

Centipeda Cunninghami, F.v.M. "Sneeze Weed." Native.

Gnaphalium japonicum, Thunb. "Japanese Cud-weed." Native.

Cotula coronopifolia, L. "Water Buttons." Native.

Helichrysum scorpioides, Lab. Native.

Leptorrhynchus squamatus, Lessing. Native.

Calocephalus lacteus, Lessing. Native.

Various Orders.

Ranunculus muricatus, L. "Sharp-pointed Crowfoot." (Ranunculaceae.) Alien.

Plantago lanceolata, L. "Ribwort Plantain." (Plantaginaceae.) Alien.

Bartsia latifolia, Sibth. & Sm. "Common Bartsia." (Scrophulariaceae.) Alien.

Acaena sanguisorbae, Vahl. "Bidgee Widgee." (Rosaceae.) Native.

Lythrum Hyssopifolia, L. "Small Loosestrife." (Lythrariaceae.) Native.

Epilobium junceum, Forst. "Hairy Willowherb." (Onagraceae.) Native.

Myriophyllum elatinoides, Gaud. "Coarse Water Milfoil." (Haloragaceae.) Native.

Erythraea australia, R. Br. "Austral Centaury." (Gentianeae.) Native.

Villarsia reniformis, R. Br. (Gentianeae.) Native.

Alternanthera nodiflora, R. Br. "Joyweed." (Amarantaceae.) Native.

Polygonum prostratum, R. Br. "Trailing Knotweed. (Polygonaceae.) Native.

Rumex Brownii, Camp. "Swamp Dock." (Polygonaceae.) Native.

Prunella vulgaris, DC. "Selfheal." (Labiatae.) Native.

Mentha Pulegium, L. "Pennyroyal." (Latiatae.) Alien.

Asperula oligantha, F.v.M. "Common Woodruff." (Rubiaceae.) Native.

Gratiola peruviana, L. "Peruvian Booklime." (Scrophulariceae.) Native.

Triglochin procera, R. Br. "Giant Arrow Grass." (Naiadaceae.) Native.

Anagallis arvensis, L. "Pimpernel." (Primulaceae.) Alien.

Myoporum insulare, R. Br. "Boobialla." (Myoporineae.) Native.

Lobelia pratioides, Benth. (Campanulaceae; Lobeliaceae.) Native.

Isotoma fluviatilis, F.v.M. "Swamp Isotoma," Campanulaceae. (Lobeliaceae.) Native.

Among the plants collected were the following poisonous, suspected poisonous, or injurious plants:—

1. Anagallis arvensis, L. "Pimpernel." Alien.
2. Calocephalus lacteus, Less. Native.
3. Centipeda Cunninghami, F.v.M. "Sneeze Weed." Native.
4. Isotoma fluviatilis, F.v.M. "Swamp Isotoma." Native.
5. Lobelia pratioides, Benth. Native.
6. Myoporum insulare, R. Br. "Boobialla." Native.
7. Ranunculus muricatus, L. "Sharp-pointed Crowfoot." Alien.
8. Senecio, lautus, Soland. Native.

Of these, *Calocephalus lacteus*, *Centipeda Cunninghami*, *Myoporum insulare* and *Senecio lautus* are merely suspected poisonous plants. The *Ranunculus* is generally stated to be feebly poisonous

when fresh. If the *Senecio* were poisonous, it would probably act like the ordinary ragwort; that is, it would slowly bring on hepatic cirrhosis as described by Professor Gilruth. The *Isotoma* and the *Lobelia* are undoubtedly poisonous, but seem to affect sheep more readily than larger stock, possibly because they are apt to eat relatively more. *Anagallis arvensis*, "The Pimpernel," was recently responsible for the death of a large number of sheep at Lilydale, the round pods of the plant being found in their stomachs in large quantities. This plant is a naturalised alien, which has rapidly spread over Victoria, and is one of those plants which it is almost impossible to suppress. It appears to act as a narcotic poison. It frequently remains green for a time when the grass is dying off, and this may attract the stock to eat it in large quantities if it is abundant. I suggest as the next stage in the inquiry, that the stomachs of the stock affected by the "disease," be examined for the presence of the plants marked 1 to 8, and particularly for numbers 1, 4 and 5.